ON INFANTILE PARALYSIS

JULIUS ALTHAUS. M.D

With the author's compliments.

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AND

SOME ALLIED DISEASES OF THE SPINAL CORD:

THEIR DIAGNOSIS AND TREATMENT.

BEING

AN ESSAY,

To which the Silver Medal of the Medical Society of London was awarded, on March 8, 1878.

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BY

JULIUS ALTHAUS, M.D., M.R.C.P. LOND.,
Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park.

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This paper was read before the Medical Society of London, on February 11th, 1878, and is now published in a separate form, the subject being one of considerable scientific and practical importance, and not having been fully understood up to the present time.

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ON INFANTILE PARALYSIS.

ALTHOUGH Ollivier's large and important work on diseases of the spinal cord appeared as long ago as 1824, yet it may be truly said that a more precise knowledge of the affections to which that organ is liable, is of very recent date; and there are now few departments of pathology in which so much remains to be accomplished as in the one to which I purpose to draw attention in this paper. Indeed, the more we have come to know of it, the more abundant appears the crop of fresh problems which spring up at every step, and for which we are as yet totally unable to offer a satisfactory solution. With regard to the functions of the cord in health, there was more agreement amongst physiologists thirty years ago than there is at the present day. It is true that we are now much better acquainted with certain functions of the grey matter in the centre of the cord, and of the white posterior columns, than was formerly the case; yet we seem, after a number of ingenious experimental investigations, more in the dark than ever

concerning the function of the white anterior columns. Again, while Pflüger has, by most able researches and acute reasoning, attempted to vindicate for the upper portion of the cord, certain functions which we have hitherto been in the habit of looking upon as purely cerebral or mental, Goltz has in almost as masterly a manner gainsaid all these conclusions. Much, no doubt, is known about the cord as a centre of sensation, motion, reflex action, co-ordination, and its influence on the movements of the bladder, rectum, and the male and female organs of generation; yet we are still unacquainted with the mode of its action on the various secretions, more especially of the salivary glands, the womb, ovaries, kidneys, and testicles, to all of which the cord appears to have an intimate relation.

The same considerations apply to the department of normal and pathological histology of the cord, on which so much light has been shed by the researches of Lockhart Clarke, Deiters, Frommann, Charcot, and others; for even at the present moment so elementary and important a question as that of the intimate structure of the neuroglia, or cementing tissue of the nervous matter in health, is in dispute. Since Virchow's researches on this point were first made known, the microscopical characters of the neuroglia have been a favourite subject of study on the part of the foremost microscopists in Germany,

England and France; and according to Boll's most recent researches it appears to consist of multipolar connective tissue cells, with numberless fine processes and nuclei. If we turn to pathological histology, there is as yet much divergence of opinion as to what should be considered inflammation, and what, on the other hand, secondary degeneration, arising not from an irritative process, but from failure of the nutritive or trophic action of the nerve-cells on the nerve-fibres. Certain pathological appearances, such as softening, disintegration, varicose nerve-sheaths, etc., may be artificially produced by post-mortem changes, unless the specimens be kept in an ice-chamber, or at least at a temperature very little above freezing-point; and the very application of chromic acid, intended for the preservation of the parts, has sometimes led to errors when the solution used was too strong, in consequence of which the peripheral portions of the specimens hardened too rapidly, and became a barrier against the further penetration of the acid, leaving the central parts unaffected, and therefore liable to decomposition.

In experimental pathology, some good and suggestive work has been done, and which promises, if continued and enlarged, to give us a clearer insight into the mode of production of the structural changes which occur in diseases of the cord. Myelitis was first experimentally produced by

Hayem and Liouville, who injected a solution of iodine in glycerine into the substance of the organ. Leyden subsequently employed for the same purpose, the liquor arsenicalis of the pharmacopœia, of which he injected from ten to twenty drops. This produced a hyper-acute purulent inflammation, resulting in extensive softening and suppuration, with effusion of blood into the tissue of the cord and beneath its membranes. In some cases, the whole marrow at and near the place of injection was changed into pus, to the extent of an inch and even more, while at other times small abscesses were found in the midst of softened matter. Microscopically, the first signs were swelling of the nuclei of the neuroglia, the nerve-fibres and cylinders-axis, all of which subsequently perished. The inflammation proved to be most severe at and near the place of injection, but was seen to spread considerably beyond it, both upwards and downwards, and to diminish in intensity as the distance increased. In the more remote parts there was no longer any suppuration, but the ordinary signs of acute myelitis presented themselves, viz., red and yellow softening, corresponding microscopically to dilatation of the arterioles and small veins, rupture of the capillary vessels with effusion of blood, swelling of the fibres of the neuroglia, nerve-fibres, cylinders-axis and ganglionic cells, and subsequent fatty degeneration and absorption of the whole

mass. At a further distance from the place of injection, small disseminated areas of inflammation, without softening, and recognisable by the microscope only, were discovered. The general result of these experiments was therefore that *myelitis is essentially the same disease*, whether it leads to suppuration, softening or slight disintegration, and that it only varies in degree of intensity.

Dr. Hamilton has worked at the same subject in the laboratory of Professor Stricker, of Vienna, but employed a somewhat less violent method than Leyden. Having laid the cord of narcotised cats bare in the upper lumbar region, he passed a thread through the organ for about an inch longitudinally, tied the ends, closed the wound, and then left the animals for forty-eight hours, so as to allow inflammation to become established. On examining the cord after death, the tissue was found to be mechanically broken down, and blood extravasated, at the seat of the lesion caused by the thread. The true inflammatory area, however, was not there, but at some distance from it in the surrounding parts. In some cases the whole extent of the transverse section of the cord was inflamed, but generally the change was most marked in the anterior columns. The microscope showed that the nerve-sheaths had undergone distension and attenuation; the axis-cylinders were swollen and divided, colloid bodies being formed from them, which were sub-

sequently transformed into pus-corpuscles. The nerve-cells had undergone œdematous degeneration, the cell-substance having been converted into a molecular mass, in which the nucleus remained visible for some time, but finally likewise passed through a similar change. The neuroglia was not so much altered as might have been expected, yet in many places its protoplasmic nuclei were seen to be much more abundant than they are in sections taken from the normal portions of the same cord.

Feinberg has experimentally produced myelitis in a rabbit, by plying its hind-legs, which were shaved, with ether-spray for half an hour. The animal became anæsthetic and paralysed, and continued in that condition for about two hours, after which time it recovered. Six weeks afterwards, however, myelitis was developed, which proved fatal. Myelitis could also be produced by cauterising the sciatic nerve with a stick of potash. All the animals thus experimented upon, died within a month of the operation. The parts between the place of cauterisation and the cord were found in their normal condition; the membranes were likewise healthy, but the substance of the cord, more especially the central grey matter, was softened. Irritation of peripheral nerves may, therefore, be transmitted to the cord, without inflammation of the intervening structures, and it is probable that this influence is exercised by means of the vaso-

motor system of nerves, causing at first contraction, and afterwards paralytic dilatation of the blood-vessels.

While it therefore appears, from the instances to which I have just drawn attention, that our knowledge in all the different branches of this subject is still fragmentary, yet no one who has watched the scientific movement of the last few years, can deny that great progress towards a better comprehension of it has been made; and I now purpose to show how far this holds good for that important portion of the spinal cord which is known as the *anterior cornua* of the grey matter.

The anterior horns are those broad and rounded processes of the central grey matter from where the anterior or motor roots of the spinal nerves emerge; while the posterior horns are those more pointed and attenuated processes from where the posterior or sentient roots originate. A central band unites the anterior with the posterior cornua forming the gelatinous substance of Rolando, while Clarke's vesicular columns are situated on the boundary line between anterior and posterior horns, in the dorsal portion of the cord.

The intimate structure of the anterior cornua is now tolerably well ascertained. Their chief histological elements consist of multipolar ganglion cells of considerable size, some of which are visible to the naked eye, when the cord has been treated with

a solution of carmine, as small points of a bright red colour, carmine having the property first pointed out by Lockhart Clarke, of colouring only the ganglionic cells with their processes, and the neuroglia, but not the nerve-fibres, which cannot absorb the colouring material. The cells of the posterior horns are much smaller, and can only be recognised by employing magnifying power; while cells of intermediate size between the two are found in Clarke's vesicular columns. These cells lie in the grey matter in peculiarly arranged groups, of which Goll has distinguished as many as twelve, while Kölliker and Leyden assume only five. No doubt the arrangement and grouping of these ganglionic masses is intimately connected with difference of function. Osjannikoff and Jacubowitsch have endeavoured to prove that the large anterior cells are strictly motor, the smaller vesicular ones of Clarke's columns sentient, and the smallest posterior ones sympathetic or vasomotor; while Duchenne and Joffroy consider that only a portion of the anterior cells are motor, and another portion of them trophic, that is, regulating the nutrition of the parts under their influence—a hypothesis which is largely supported by facts in pathological histology and clinical medicine.

In some portions of the cord, the ganglion cells are much more numerous than in others. Thus Goll found 140 in that part of the anterior horns which

corresponds to the cervical enlargement at the exit of the sixth cervical nerve, where this assumes its greatest development; seventy-seven in the part corresponding to the fourth; forty-two in that answering to the third; thirty-eight corresponding to the eighth; and twenty-eight in that nearest the first cervical nerve. These cells are again very numerous in the lumbar enlargement of the organ, but scanty in its dorsal portion. In fact both the cervical and lumbar enlargements consist almost exclusively of an accumulation of grey matter in the anterior horns.

The cells enclose a large nucleus with a bright nucleolus, and heaps of pigmentary granules. Their shape is angular, stellated or spindle-like. Deiters has discovered that two different kinds of processes branch off from these cells, viz., firstly, numerous so-called protoplasmic processes, which radiate and ramify in all directions, and ultimately form a very fine network; and secondly, one single so-called nerve-process, or cylinder-axis, which is distinguished from the protoplasmic processes by its darker and better defined contours, and its greater resistance to test solutions. The nerve-process does not ramify like the others, but remains undivided, and after proceeding for some distance, becomes surrounded with a sheath, and is changed into a nerve-fibre, which has in many instances been followed up into the anterior nerve-roots, to the fibres of which it becomes adjoined.

The number of protoplasmic processes varies. Where they are numerous, the cells are called multipolar; where there are only two, we speak of bipolar cells; but the so-called apolar cell, without any process, is now generally considered to be an artificial production, owing to the processes having been destroyed in preparing the specimens. The processes being assumed to propagate and transmit the nerve-force engendered in the cells, an apolar cell would indeed have no *raison d'être.*

Next to the cells we find in the grey matter very fine nerve-fibres, some of which contain myeline, and others not. Those containing that substance are a good deal finer than the corresponding fibres in the white matter of the posterior and antero-lateral columns of the cord; while such as are without myeline belong exclusively to the grey matter, in which they form a very fine network, within which the finest nerve-fibres communicate most abundantly with the protoplasmic processes of the cells; cells and fibres being surrounded, and, as it were, cemented by the neuroglia.

While carmine colours the cells and neuroglia, but leaves the fibres unaltered, chromic acid and its congeners harden the parts, so that it becomes easy to make sections, and likewise allow us to distinguish at a glance healthy from diseased tissue, since the former assumes a dark yellow colour, while the latter remains light. The first to suggest

the use of chromic acid for this purpose was Prof. Hannover, of Copenhagen; Lockhart Clarke afterwards substituted bichromate of potash for it, while Gerlach prefers bichromate of ammonia. For rendering the specimens transparent, glycerine, oil of cloves, benzine, turpentine and creosote are generally employed.

Clinically the principal diseases in which the characteristic lesion may, during life, be supposed, and is after death discovered, in the anterior cornua, are infantile paralysis, certain forms of paraplegia, without simultaneous loss of sensation, and coming on either suddenly or gradually, and progressive muscular atrophy.

I. *Infantile Paralysis.*

Infantile paralysis is produced by a diffuse acute inflammation of the anterior horns, which is generally most intense in the cervical or lumbar enlargement, but may also affect the dorsal portion of the cord. It proves but rarely fatal within a few days or weeks from its invasion. Occasionally there may be no general symptoms at all, but the child is put to bed apparently in perfect health, is found next morning paralysed in a limb or part of a limb, and ails otherwise nothing at all. This mostly occurs in hearty, well-developed children, whose

nervous system has already some power of resistance, and where the inflammation occupies only a small area, so as to cause paralysis limited to part of a limb, or only some sets of muscles. In a much larger number of cases, however, there is a good deal of constitutional disturbance, viz., high fever preceded by general malaise, loss of appetite, and pain in the head, as evidenced by screaming, and by the child pressing the head into the pillow. One or several fits of eclampsia then take place, after which the paralysis is noticed. This latter does not occur as quickly as in apoplexy from cerebral hæmorrhage, but nevertheless very rapidly; it mostly reaches its maximum within a few hours, and has no progressive character whatever. The fever abates after a time, and the general condition becomes satisfactory, but the paralysis remains. In exceptional cases there are several attacks of it, so that first one limb is paralysed, after which the child appears to improve, but relapses a few days afterwards into a feverish state, when another limb is found affected, etc. A fatal termination of the illness is exceedingly rare, and there are no post-mortem records where the child has not been paralysed for at least two months; yet I believe that certain cases of infantile eclampsia, which proves so largely fatal to children, are really instances of severe inflammation of the anterior cornua of the cord, more especially when occurring

in weak and sickly children, and when affecting the entire extent of the cord. In such cases the principal symptoms are a succession of convulsive seizures, and hyperpyrexia; the child appears paralysed after the fits, sinks into a comatose condition, and dies from asphyxia or exhaustion of the nervous centres.

In cases which have proved fatal within a few months from the invasion of the disease, simple inspection of the cord by the naked eye teaches us little or nothing. Rilliet and Barthez, the authors of the most able and comprehensive work on the diseases of children, were thus led to express the opinion that there was no distinctive anatomical lesion connected with infantile paralysis. In Germany, however, Heine, who was extremely familiar with these cases from having watched large numbers of them in the orthopædic institution with which he was connected, was by clinical observation led to the view that the disease was of spinal origin. The first microscopical observations of specimens of the cord were made by Cornil, Prévost, and Vulpian, to which were soon afterwards added those of Lockhart Clarke, Charcot, Joffroy, and many others. The general result which has thus been obtained is, that infantile paralysis is an extremely acute myelitis of moderate intensity, which is either diffuse or occurs in circumscribed areas, and affects more particularly the cervical

and lumbar enlargements of the cord. The inflammation being generally confined to the anterior grey matter, Kussmaul has proposed to call the disease anterior polio-myelitis, from πολιος, grey, and μυελος, marrow; and as this term appears to be a very appropriate one, I propose to adopt it.

The right leg being most frequently the seat of infantile paralysis, the right anterior horn of the lumbar enlargement is chiefly subject to this inflammation. Where only one limb suffers, we speak of monoplegia; but there may also be hemiplegia, paraplegia, and crossed paralysis, in which latter form the arm of one side, and the leg of the opposite side are affected. The paralysis is, however, not necessarily confined to the limbs, but may affect the body, more especially the muscles of the back; in such cases we find that the children are unable to sit up; they are apt to fall forwards, more rarely backwards, and the spine assumes a more or less considerable degree of curvature. There is never any affection of the head or of the cranial nerves, which renders it evident that the disease is confined to the spinal cord, and does not proceed as high up as the medulla oblongata. Where the muscles of the body have been paralysed during life, changes in the dorsal cord are discovered post-mortem, while, where the extremities had suffered, the cervical and lumbar enlargement are found diseased.

The question whether polio-myelitis affects primarily the ganglionic cells of the anterior cornua, or whether the neuroglia, or interstitial cementing tissue, is first affected, and softening and atrophy of the ganglionic cells is subsequent thereon—in other words, *whether polio-myelitis is parenchymatous or interstitial*—is not yet settled. Charcot and others support the former view, while Roth and others are in favour of the latter. Dujardin, however, has rendered it probable that both tissues become inflamed at the same time, and that the myelitis is therefore parenchymatous as well as interstitial. The microscope shows indeed the whole structure to be inflamed; the large multipolar cells together with the nerve-fibres and cylinders-axis are more or less extensively destroyed; there is proliferation of nuclei, and connective tissue, and hyperæmia of the blood-vessels. The neighbouring tissues are generally normal, as the inflammation has no tendency to spread either to the posterior cornua, or to the antero-lateral columns; and it is only in cases where there have been complications during life, and where the true clinical aspect of infantile paralysis has therefore been somewhat obscured, that changes in other portions of the organ have been discovered.

I have said that death at an early period of infantile paralysis is exceedingly rare; and most cases, therefore, which have been ultimately

examined on the post-mortem table have proved fatal at much later periods of life, owing to incidental diseases which had no connection with the palsy. One case is on record where the autopsy took place upwards of sixty years after the occurrence of the paralysis. In such instances the post-mortem appearances are of course somewhat different from those which obtain in recent cases; but the general result is the same. The naked eye perceives a wasting of the anterior cornua; the antero-lateral columns are smaller than in health, but the posterior cornua and columns are unaffected. If specimens of the diseased parts are microscopically examined, it is seen that in lieu of the cells and fibres which constitute the principal parts of the anterior horns in health, there is connective tissue of various periods of age, some young and soft, some tough and old, while amyloid corpuscles, which are rarely absent in old destructive lesions of the cord, are found in considerable quantities. If any cells are left, they are seen to be in different stages of shrinking and disintegration. Clarke's vesicular columns are generally in their normal state. The peripheral nerves which have been paralysed during life, are wasted, and the connective tissue and nuclei appear increased. The muscles are in a state of degenerative atrophy. At an earlier stage of the complaint they appear pale, soft, and attenuated; the primitive fibres are

small, the stripes indistinct, the nuclei proliferated, the interstitial tissue hypertrophied, and the blood-vessels thickened. Later on the muscles assume a greyish tinge, and the fibres are interspersed with whitish connective tissue and layers of yellow fat. Ultimately the quantity of fat which is deposited, may become so abundant that the entire muscle is replaced by it, and there may be actually more bulk than there would be if the muscle were in its proper condition. Other portions of the muscles may undergo a kind of fibrous sclerosis, without development of adipose tissue. The tendons are smaller and thinner than in health; the bones are shorter and narrower; their protuberances are not properly developed, the cortex is thin and fragile, the medullary layer enlarged, the marrow more abundant. The joints are relaxed, the ligaments loose and flabby, the cartilages attenuated. The arteries are somewhat wasted, but the skin over these parts is generally normal.

Anterior polio-myelitis of children is distinguished from other forms of spinal paralysis by there being no affection of sensibility, which remains perfectly normal; no paralysis of the sphincters, which continue to act properly throughout the course of the disease; and no tendency to decubitus. It is true that some of these little patients suffer from nocturnal enuresis, but there is no real incontinence of urine. *It is therefore exclusively motor paralysis,*

followed by muscular atrophy, to which is frequently added contraction and deformity.

The mere existence of the paralysis is easily recognised, without having recourse to the more minute methods of examination which are necessary for determining the exact condition of the paralysed parts. A limb, or portion of the limb, or several limbs, remain motionless when the rest of the body is moved about. The affected limb is flabby and loose, it dangles about, slips away, does not resist passive movements which are impressed upon it, and assumes any position which is given to it by gravitation. The muscles feel flabby to the hand, and this increases within the next few weeks, as wasting of the muscular substance, which is no longer connected with its centre of nutrition, sets in and becomes more or less fully established. The joints become so loose that incomplete dislocation is frequently produced. The wasting of the muscles is sometimes hidden from the eye or the hand by a considerable accumulation of fat, more especially in the thigh, while it is more easily recognised in the shoulder, arm, and leg. The ends of the extremities, such as the knees, toes, fingers and elbows, may be slightly cyanotic, somewhat swollen, and one or two degrees colder than the corresponding ones of the other side; and they are generally covered by a cold clammy perspiration, which is absent from the healthy limbs.

The exact state of the nerves and muscles can, during life, only be recognised by using electricity; and for this purpose both forms of current, the induced or faradic, and the continuous or voltaic currents are necessary, as the nerves and muscles respond to both forms of current in a different manner; and from the way in which the response takes place, conclusions may be drawn as to the more or less altered condition of the nerves and muscles. The induced or faradic current should be applied first to the motor nerves, when we speak of indirect muscular faradisation; and secondly, to the tissue of the muscles themselves, which is called direct muscular faradisation. In the former of these two modes, the negative pole or cathode which has a more powerful influence than the positive pole or anode, is applied to the motor nerve, while the other pole is placed to some point at a distance, for which the sternum generally is chosen. In health, such a proceeding causes contractions of all the muscles which are under the influence of the motor nerve that is faradised. The degree of these contractions varies according to the force of the current used, so that a feeble powe causes slight, a moderate force moderate contractions, while a powerful current produces true tetanus of the muscles. This effect of the induced urrent is entirely independent of the will of the person experimented upon, and irresistible, provided

a current of some power is used. Direct muscular faradisation is performed by placing both electrodes on the belly of the muscle which is to be examined and is quite local in its effects.

Where infantile paralysis is complete, and has lasted for some time, we find that this faradic response of the muscles is totally absent, even where a considerable force of current is employed, and whether we use direct or indirect muscular faradisation. The muscle appears completely dead and unexcitable to this form of electricity; while in other forms of paralysis, more especially in hemiplegia from cerebral hæmorrhage, or from softening by embolism and thrombosis, the paralysed muscles generally respond equally well to faradisation as the healthy ones, and continue to do so for years after the paralysis has become established. We may, therefore, by this means alone distinguish infantile paralysis from cerebral palsy. Where the loss of power is incomplete, the faradic excitability of the muscles is not entirely gone, but more or less considerably diminished; and this can be accurately ascertained by comparing the corresponding muscles of the healthy limb with those of the paralysed one, when it is seen that an electric force which is capable of producing a decided effect on the healthy muscles, will remain ineffectual in the diseased muscles; and that the force must be considerably increased before any effect in the latter is witnessed.

Duchenne has shown that the faradic excitability of the nerves and muscles begins to diminish at a very early period of the disease, viz., on the third, fourth, or fifth day after the palsy has become established, and that it has completely vanished by the seventh day, or at the latest in the second week.

Where this loss of faradic excitability is complete, the muscles, unless specific treatment be immediately adopted, generally remain permanently paralysed; while where there is only diminution but no absolute loss in the second week, the muscles may be expected to recover, and that the more rapidly and completely, the less this faradic excitability was diminished.

The continuous or voltaic current, when passing through a nerve or muscle for some time without opening or closing the circuit, produces no apparent effect, while any variation in the current, such as making or breaking it, will cause a muscular response. This latter takes place in a peculiar manner in the healthy subject, circumstances being somewhat more complicated than with faradism. With faradisation the effect of the two poles is identical, except that the negative is more powerful than the positive; but with the continuous current there is not merely a difference in degree, but also in kind, as far as the two poles are concerned. In using it for the purpose of ascertaining the condition of the nerves and muscles, the cathode and

anode are alternately placed on the nerve, while the other pole rests on the sternum, or some other place at a distance. The current is then alternately made and broken, when it is seen that the cathode acts chiefly on making, and the anode chiefly on breaking, and that the cathode is more powerful than the anode. Thus when a feeble current is used, there is only a response on making with the cathode, but no effect takes place on breaking with the cathode, nor whether we make or break it with the anode. When the force of the current is increased, the response on making with the cathode becomes more powerful; there is no effect on breaking with the same; but the anode causes slight contractions on making as well as on breaking, the latter effect being the more powerful one. Finally, with the highest degree of current force, there is strong tetanus on making with the cathode; a second somewhat more feeble effect on breaking with the anode, a third still weaker action on making with the anode, and a fourth extremely slight response on breaking with the cathode. The muscles respond to the continuous current in the same manner as the nerves, and any changes in the mode in which these voltaic responses take place are pathological, and give us valuable indications, not only as to the conditions of the paralysed nerves and muscles, but also for the diagnosis, prognosis, and treatment of the affection we have to deal with.

That the voltaic excitability of the nerves and muscles is altered in infantile paralysis, was first shown by Salomon, and has since then been frequently noticed by a number of competent observers. Here a curious fact presents itself, which has long been known with regard to peripheral paralysis of the motor nerves, viz., that nerves and muscles obey totally different laws as far as the voltaic influence is concerned. The *nerves* lose voltaic excitability in the same ratio and degree as they lose their faradic response, that is to say, there is a diminution within the first few days, and total abolition in the second week. With the *muscles*, however, the case is entirely different; for while during the first week the voltaic response diminishes proportionately to the faradic excitability, it begins to rise in the second week, and then becomes exalted beyond the normal standard, so that the paralysed muscles are seen to answer to a current so feeble as to be absolutely ineffectual when applied to healthy muscles. Thus I have repeatedly seen that, when the healthy muscles responded only to twenty-five or thirty pairs of the battery, the paralysed ones answered to ten or fifteen pairs. Apart from this, however, the character of the response is altered. For while the healthy muscle gives a quick and short response, and that only on making and breaking, but not while the circuit remains closed, the paralysed one answers sluggishly, in a

drawling manner, and remains in a kind of continuous contraction during the whole time that the current continues to act. The normal response to the positive and negative pole is likewise altered, so that the cathode, which generally predominates over the anode, loses, and the anode gains, in influence; we find therefore that the effect of making with the anode becomes stronger than that of making with the cathode. At the same time the effect of breaking with the cathode, which in health is of the very slightest, is increased, so that it becomes after a time equal or even superior to breaking with the anode. This condition continues for about a month, or more, after which the response on making with both poles disappears; and this is then gradually succeeded by a decided fall in the galvano-muscular response altogether. This fall continues, so that in order to obtain any response at all, the current-force has to be considerably increased, two or three months after the commencement of the palsy. Ultimately any degree of voltaic power which it is possible to use, produces either no effect at all, or only a very slight one, on making with the cathode. Even in cases where, ultimately, recovery takes place, galvano-muscular excitability continues feeble for a considerable time, and the recovery of volitional power begins not unfrequently at a time when both faradisation and galvanisa-

tion are still quite, or at least nearly, ineffectual. Some years after the paralysis has become established, the voltaic current is the only means of showing that muscular tissue is still in existence, a sluggish response being obtained by a considerable degree of galvanic force, principally on making with the cathode.

I cannot in this place enter into a full explanation of the singular and interesting phenomena which I have just described; suffice it to say that they are to a great extent accounted for by the differences in the duration of the currents which are used in these investigations. The shorter the duration, the less is the effect. This is the reason why faradism, which consists of a very quick succession of instantaneous currents, has no effect at all; and the voltaic current likewise is more effectual in proportion to the length of its action. A feeble voltaic current, which acts for a considerable time, has for this reason more effect than a powerful one which acts only for an instant. Pathologically the phenomena which I have just described, correspond closely to those degenerative changes which are going on in the nerves and muscles, when these have been separated from their nutritive centres in the cornua of the cord; and it is thus shown that destruction of these centres has the same effect on the nerves and muscles which are under their influence, as is produced by a break

in the continuity between the centre and the peripheral nerve, as, for instance, in peripheral paralysis owing to injury of the nerve. It is, therefore, no longer true, as was formerly believed, that the presence of the galvano and farado-muscular phenomena just described, was characteristic for peripheral paralysis; but the conclusion is inevitable that when they present themselves, we have to do either with destruction of the nutritive centres, or with separation of the peripheral parts from these centres; and the diagnosis between separation and destruction cannot be made by electricity, but must be arrived at by a consideration of the general clinical features of the cases under examination. For the electrical phenomena just described, I propose the term of "*wasting-test.*"

II. *Anterior Myelitis of Adults.*

Does infantile paralysis ever occur in adults? This might by some be considered a Hibernian question, but it is one which has often occupied the attention of pathologists. Some observers have denied that it occurs after eleven years of age, while a number of others have described cases of myelitis and of post-febrile paralysis in adults, which show the greatest possible analogy with infantile paralysis. As such cases are not very

common, I will now give the details of two such, which are at present under my care. In one of them the disease affected the anterior horns of the lumbar, and in the other the same portion of the cervical enlargement.

The first of these patients was an officer in the army, aged 31, single, of healthy parents and temperate habits. He had entered the army in 1864, had from that time until 1869 been stationed in the Northern provinces of India, and afterwards at various places in England. In 1874 he was sent to Malta, where he remained three years. At that time he was in the habit of taking violent exercise, as he found himself getting stout. He used to ride much on horseback, and played at rackets for two or three hours daily, which threw him into a violent perspiration. On June 25th, 1877, he was out taking exercise in a very powerful sun, and towards evening took a bath in the sea. The water felt very chilly to him, but he nevertheless remained about three-quarters of an hour in it, sometimes standing about on the shore, and then getting back into the water. On coming out at last, he felt benumbed, and had acute pain in the small of the back, and the legs; he could not recline nor sit still for even a few minutes, and felt so restless that he did not sleep at all, but kept walking about in his rooms all night. Hot fomentations relieved the pain, but only for a short time, and he was much

exhausted in the morning. On the second day the pain was not so acute; he could walk about without assistance, but the back felt stiff, and the legs ached. Towards evening of the same day he could not walk so well; he had to hold on to the shoulders of two brother-officers, and the right leg dragged. He could pass his water without difficulty. There was loss of appetite; he only took a little soup. Hot sponging relieved him, and made the limbs feel easier for a short time. At night he had an opiate, and slept for sixteen hours consecutively, perspiring profusely all the time.

On awaking on the morning of the third day, he felt exhausted, and found that his legs had become entirely powerless from the hips downwards; he could only move them with his hands; the acute pain was gone, a dull aching only being felt. The back and loins felt very stiff, and he could not turn over in bed, showing paralysis of the muscles of the loins. The legs were tender to the touch, and felt sore when handled. He had to strain a little in passing his water, and the bowels were confined. The loss of appetite continued. The patient was now blistered and leeched in the back. The temperature, which had previously not been taken, was now found to be 104°, with the thermometer placed under the tongue. The next night he slept a little, the restlessness being relieved by his position being shifted frequently. There was no feeling of tight-

ness round the lower portion of the body, nor any loss of sensation.

On the fourth day there was no acute pain, but the dull aching in the back and legs continued. The patient was put into an easy chair and felt better. The bowels were relieved by an enema, and the water was passed after some delay, the stream appearing fairly strong. The appetite was improved, the temperature 103°. The day and night registrations of the thermometer varied but little. The legs were rubbed, which made them comfortable, and they became red under the friction. There was no priapism at this or at any other time.

The succeeding days were much the same. The thermometer gradually went down to 101°, and remained at this figure for three weeks more. The general condition improved, the bowels acted regularly, and the water could be passed at all times. The appetite was better, and the patient slept longer without requiring to have his position changed so often, being easier on the back than on the side. He generally spent the day sitting in an easy-chair, which did not fatigue him. The legs, however, now began to waste considerably.

In about three weeks the temperature had fallen to 98°, and the patient was then given solid food. The general condition was now satisfactory, and he began to move the toes of the left foot a little. Strychnia was injected subcutaneously in the inner

part of the thighs, and rubbing and magneto-electricity were used for about a fortnight. He was invalided in the commencement of September, and left Malta. He went to Guernsey, where he was treated with strychnia three times daily, and the continuous current; with the effect that the muscles developed better and became firmer; and in December last he came to London to place himself under my care.

On examining him on December 13th, I found that there was complete paralysis of motion in both lower extremities, from the hips downwards. The muscles of the loins were not paralysed, as the patient had no difficulty in turning over in bed; but there was no movement whatever in the ankle-joints, the knees and the hips, while the toes of the left foot could be slightly flexed. The wasting-test was well marked. Faradisation of the nerves and muscles of the lower extremities did not produce any response at all, even if the power of the current was increased to the maximum strength given by the double-celled coil of Stöhrer's apparatus, and which caused an almost intolerable sensation of pricking and burning; yet not the slightest fibrillary twitches occurred in any of the muscles acted upon, whether direct or indirect faradisation was used. The continuous voltaic current had no influence on the nerves, but when applied directly to the muscular substance, caused

sluggish contractions, more particularly in the left leg. Making with the anode had the most effect, making with the cathode less, and breaking with either poles was quite ineffectual. There was no muscular rigidity anywhere, all the muscles being completely flabby and relaxed, and offering no resistance to passive movements impressed upon them. They had a doughy feel, and as there was not much wasting in the limbs, a transformation of some portion of muscular tissue into fat could be assumed with a high degree of probability.

Sensation was perfectly normal, there being neither pain nor paresthesia nor anæsthesia. Pressure on, and percussion of the spine did not show any tender points; a hot sponge could be carried along the back, without giving rise to a feeling of soreness; and faradisation and galvanisation of the spine showed it to be in its ordinary condition of sensibility. The senses of touch, of temperature, of locality, and all the other varieties of sensation in the lower extremities were likewise normal.

Reflex excitability, on the other hand, was completely abolished. Tickling the soles and the knees, and irritation of the inner surface of the thighs, was well perceived, but did not cause any movements in the paralysed limbs. Percussion of the tendon of the rectus femoris and of the tendo Achillis, remained likewise ineffectual.

The temperature of the limbs was 94° to 95°.

The skin was slightly moist, œdematous, and in parts mottled. There was no decubitus, nor had there ever been any sign of it. The sphincters were perfectly normal; the action of the bowels regular; the urine normal; nor was there any loss of expulsive power in the bladder. Sexual desire, however, which before the affection came on was keen, was lost, and no proper erections nor nocturnal emissions had occurred since the disease became developed. Digestion, respiration, and the heart's action were quite normal. There were no head symptoms.

On considering the peculiar features of the case, the diagnosis could not be doubtful. As only the motion of the lower extremities was lost, and sensation in them had not suffered; as there was no decubitus, and no affection of the bladder and rectum, the case was evidently one of acute poliomyelitis, or inflammation of the anterior cornua of the lumbar enlargement of the cord, which had led to paralysis and atrophy of the muscles of the lower extremities. The close analogy of the symptoms with those which are observed in the majority of cases of infantile paralysis, was very striking. There had been fever, the thermometer having run up to 104°, and remaining for more than three weeks above the normal average. There had, however, never been any head symptoms, which is explained by the circumstance that the brain of

adults offers more resistance to morbid influences of this kind than that of children; and this is more particularly so where the seat of the disease is at a considerable distance from the brain, as in the lumbar enlargement. Where the upper portion of the cord is affected, cerebral symptoms may make their appearance in the adult, as I shall presently show by another case. In this instance there had only been general malaise, with pain and tenderness of the loins and lower extremities, restlessness, and complete loss of appetite. As is so well marked in infantile paralysis, the loss of power had set in rapidly, there having been paresis or incomplete paralysis on the second, and complete paralysis on the third day, with total relaxation of the muscles. From that time the state of motion had not perceptibly varied. The bladder was only to the very slightest extent affected on the third day of the disease, but rapidly recovered its full power. The fact that there was at no time any muscular rigidity, explains why no deformity worth speaking of took place. In the adult, deformity is altogether less likely to occur than in children, because the joints and ligaments are firmer, and the growth of the bones is finished at the time of the invasion of the disease; and deformities occur as a general rule only where one set of muscles is more paralysed than another, or where one set has escaped the lesion altogether.

I now proceed to describe a case of acute anterior polio-myelitis affecting the *cervical* enlargement of the spinal cord.

M. M., a Scotch gentleman, aged thirty-four, single, of no regular occupation, came under my care in January, 1876, and gave me the following history :—

'In November, 1864, being then an undergraduate at Edinburgh, in good health, and studying hard, he attended a crowded meeting in the music hall in that city, during which he perspired profusely. When he came out of doors, it was snowing, with a keen northerly wind blowing. He dined, and then walked out again for about an hour. Before returning home, he felt very chilly; two hours later he perceived pain and stiffness in the back of the neck, which increased when he attempted to move his head. He gradually became more chilly and drowsy, and tried hard to shake it off, so as to be enabled to study, but to no purpose. At bed-time, the skin being hot and dry, he took some hot whisky and water, but continued feverish, and lay tossing about in bed all night, very restless, and unable to go to sleep. Next morning he got up and went to his class, but felt so drowsy and languid that he was obliged to return home. That afternoon he took a dose of Epsom salts, and having during the following night to traverse long windy passages to the closet, he took a second chill. The two following days he was up, but did not go out, as the

excessive drowsiness and languor continued without abatement. During the night of the fourth day, he got out of bed for a drink, but after making a few steps his knees gave way quite suddenly, and he fell heavily on his back. A friend with whom he was lodging, had to assist him to get up, and with some difficulty put him into bed. He now perceived a difficulty in passing his water, but ultimately succeeded in doing so. Next morning he found that he was entirely helpless, the paralysis having extended to both upper extremities. The left side of the body was now completely paralysed, while on the right he could just move the fingers and toes a little. All this time there was no pain, nor loss of sensation, and the fever presently left him. The temperature was not taken at any time during the progress of the case. He was given antimonial wine, and fell into a profuse perspiration, which continued without intermission for about a fortnight. The drowsiness gradually left him, the head appeared quite unaffected, and the general health was good, for with the exception of the loss of power, he felt about a month after the commencement of the disease, as well as ever he did in his life. There was however complete paralysis of the left and nearly complete loss of power in the right side. He could not even move his head on the pillow and for six months was not taken out of bed.

There was never any tendency to decubitus. The condition of the bladder varied; the patient being sometimes unable to empty the viscus, so that the catheter had to be used; while generally he could pass his water, although with some difficulty. The urine would appear never to have undergone those changes which are so marked in transverse myelitis; viz., alkaline decomposition with formation of triple phosphates, vibriones, bacteria, and muco-pus; but it would seem to have been normal throughout the disease. The bowels were sluggish, purgatives had no effect, and enemata were found necessary. The treatment at this time consisted of blistering at the back of the neck, strychnia, and the use of the magneto-electric rotatory apparatus, which was kept grinding away in his hands, and sometimes at the soles of the feet—a most unscientific and foolish application—three times daily for half an hour. The electrodes were brass tubes without handles; the machine was used for two or three months, and the patient believed it did him more harm than good. He was very little better by May of the following year, and was then taken to his home in the Highlands, where he began to improve considerably, and had in a month recovered full power over the bladder and bowels. In June he went to the Strathpeffer sulphur springs, and derived apparently some benefit from them; for when he returned, he could stand when put

on his legs, and walk a few steps without assistance.

During the following years he pursued various plans of treatment, consisting more especially of different applications of electricity, arsenic, and hydropathy; and he gradually improved, without, however, being able to attribute any very decided result to any of them.

At the time he came under my care, more than eleven years after the commencement of the disease, there were no symptoms pointing to an affection of the brain or cerebral nerves. Sensation was perfect all over the body, and the general health was good. With regard to the state of motion in the upper extremities, the patient had to a great extent recovered the power over the right arm and hand, which he could move in all directions; he could write a letter, wash and dress himself, and squeezed the dynamometer to the extent of forty-eight kilogrammes. The muscles, although not robust, were fairly nourished, and responded tolerably well to faradisation and galvanisation. The left arm was much more feeble than the right; there was some wasting of the trapezius, deltoid and serratus muscles, causing slight dislocation of the caput humeri. The biceps was very feebly developed, and the flexor muscles of the forearm were also in a state of atrophy. The muscles of the ball of the thumb, and the first interosseous muscles were greatly wasted.

In accordance with these symptoms, the patient had but little power in the left arm, which he could not raise above the horizontal line, and the hand was to a great extent useless and awkward. There was, however, a degree of faradic and galvanic response, which was in exact proportion to the general state of motion in, and nutrition of, the muscles. Reflex excitability was normal in the right, and somewhat reduced in the left arm. With regard to the lower extremities, it was found that when once fairly on his feet, he could walk with the aid of a stick for about two hundred yards. After doing that distance, however, the back became painful, and the patient got out of breath, so that he was obliged to rest. With crutches he could walk several miles. He could stand for hours without fatigue, but was always worse after sitting for a long time. He had great difficulty in turning in bed, and in undressing, and was generally unwieldy. The left leg was much more powerless than the right, and there was much greater atrophy in the muscles of the left thigh and leg. Nevertheless, what was left of these muscles responded well to galvanisation and faradisation; and there was no lack of reflex excitability in the lower limbs, both as far as skin and tendons were concerned. Apart from the unsatisfactory state of motion, the patient was in perfect health in every respect.

From this description it will be seen that the two cases of anterior polio-myelitis which I have just described, were in most respects exceedingly similar. In both the cause was evidently cold, although it does appear singular that both patients had often before been exposed to chills on a heated body without having been any the worse for it. More especially the subject of the second case was brought up inured to hardihood, and could with perfect impunity endure severe and prolonged exposure to heat, wet and cold, and was in the habit of taking cold baths to an inordinate length; and even at the present time a thorough drenching and sitting in wet clothes would not affect him. It appears, therefore, probable, that in both cases there must, at the time of the invasion of the disease, have been a state of diminished resistance of the cord to external unfavourable influences. Neither of the patients had the neurotic constitution; neither had had syphilis. In both there was fever and systemic disturbance at the commencement; in the former where the lumbar enlargement was affected, the head remained perfectly clear, there being only great restlessness; while in the latter, where the cervical enlargement suffered, there were, in addition to restlessness, great drowsiness and languor. In both there was profuse perspiration at an early period of the disease; the paralysis was quickly produced, and remained exactly the same for a con-

siderable time. Where the lumbar enlargement suffered, the bladder was hardly at all affected, while in the case of cervical polio-myelitis the bladder suffered, but recovered its tone at an early period.

There are indeed few diseases which could be confounded with acute anterior polio-myelitis. *Hæmorrhage into the spinal cord* may produce sudden paralysis, which is followed by atrophy and loss of reflex excitability; but there is an absence of fever, the invasion of the paralysis is still more sudden, as it generally comes on in a quarter of an hour, or even less; and there is anæsthesia, paralysis of the sphincters, and decubitus.

In *acute central* or *transverse myelitis* there is always anæsthesia of the skin, paralysis of the sphincters, and tendency to decubitus, by which that disease is sufficiently well distinguished from acute anterior myelitis.

Progressive muscular atrophy is very chronic in its invasion, and there is wasting of muscular tissue before the paralysis sets in. There are many other distinctive features between these two diseases, but the two I have just mentioned are quite sufficient for us to make a proper diagnosis.

Brown Séquard's *spinal hemiplegia* or *hemiparaplegia* can hardly be confounded with anterior myelitis. It is true that there is motor paralysis, affecting either the leg alone, or the arm and

leg of the same side; but while in polio-myelitis sensibility remains normal, there is in Brown-Séquard's disease hyperæsthesia on the paralysed, and anæsthesia on the opposite side, which renders the diagnosis certain.

From *cerebral hemiplegia* anterior myelitis may be distinguished by the different commencement of the paralysis; by the affections of cerebral nerves which accompany cerebral paralysis; and particularly by the circumstance that in cerebral paralysis the farado-muscular excitability remains generally unaltered, or nearly so, even years after the invasion of the disease, while in polio-myelitis it is lost in the second week.

Paralysis from obstetric operations is noticed immediately after birth, while polio-myelitis occurs very rarely before the child is twelve months old. A not unfrequent form of obstetric paralysis is that affecting the portio dura, which is caused by pressure of the blade of the forceps on the parotid gland, and the pes anserinus. It generally disappears a few days after birth, but where the pressure was severe, it may persist during life. It only becomes of importance where that branch of the nerve is paralysed which supplies the lips, as it may then interfere with sucking. In infantile paralysis, and anterior myelitis of the adult, the portio dura is never affected. The arm may also be paralysed by pressure of the forceps on the brachial plexus, which

then becomes compressed by an effusion of blood; and in such cases anæsthesia is combined with the paralysis. Pressure of the finger on the axilla, during the operation of turning, may likewise give rise to it, and cause at the same time dislocation of the caput humeri. Paralysis of the lower extremities may be produced by too forcible traction in turning, causing injury to the spinal cord. The combination with anæsthesia, and the appearance of the paralysis immediately after delivery, are sufficient to distinguish it from true infantile paralysis.

Sclerosis of the lateral columns of the cord cannot easily be confounded with polio-myelitis. It is true that there is no affection of sensibility in such cases, no decubitus, and no paralysis of the sphincters; but the invasion of the complaint is essentially chronic; and incomplete or complete paralysis, with muscular rigidity and increased reflex excitability, more especially of tendons, are the chief symptoms. Where the patient is able to walk he shows a peculiarly rigid or spastic gait, which is entirely different from the halting and lame gait of a person suffering from chronic myelitis, as well as from the jerky and unsteady walk, which is seen in locomotor ataxy. Sclerosis of the lateral columns may however coexist with wasting of the anterior cornua, and then constitutes a disease which Charcot has recently described as *lateral amyotrophic sclerosis*. This mostly begins in the upper extremities, which be-

come more or less paralysed and wasted, while the antagonists of the paralysed muscles become rigid and contracted. The consequence of this is, that the arm is held tightly to the body, the forearm flexed and pronated, and the hands and fingers strongly flexed. After this has existed for some months the disease progresses to the lower extremities, causing incomplete or complete paralysis with rigidity. There is no anæsthesia, no decubitus, no paralysis of the sphincters. After a time the rigid muscles begin likewise to waste, when the contractions disappear. Death takes place by the degeneration attacking the motor nuclei in the medulla oblongata, with consequent paralysis of the lips, tongue, pharynx, and larynx, that is, labio-glosso-pharyngeal paralysis. The chronic and progressive course of the disease is sufficient to distinguish it from polio-myelitis, the onset of which is rapid.

III. *Chronic Spinal Paralysis.*

Duchenne has described a disease which he calls *sub-acute or chronic anterior spinal paralysis.* This has some points in common with the one to which I have just drawn attention, but is sufficiently distinguished from it by its onset and progress being essentially chronic. It occurs in adults, and leads to paralysis and muscular atrophy in the legs, but

there is no fever, the paralysis is slowly developed, and it has a tendency to progress upwards, so that after a time the upper extremities become likewise involved. Anatomically it consists of chronic inflammation of the anterior cornua of the lumbar and cervical enlargement, which leads to wasting of the multipolar ganglionic cells, thickening of the coats of the blood-vessels, and overgrowth of the nuclei of the neuroglia. The anterior nerve-roots are wasted, and the muscles are atrophied or in a state of fatty degeneration. The causes of the affection are as yet obscure; cold, injury, and excesses of various descriptions are believed to give rise to it. The first symptoms are generally lassitude and fatigue in walking, and pain and stiffness in the loins and the lower extremities. After a time there is decided muscular weakness, sometimes only in one, at other times in both legs, which gradually increases, and ultimately merges into complete paralysis. Soon afterwards wasting of the muscular substance sets in, accompanied with loss of reflex excitability, while sensation remains normal. The paralysis then spreads to the upper extremities, which become awkward and clumsy, and ultimately refuse service altogether. The muscles of the back and abdomen participate occasionally in the affection, but the bladder, rectum, and sexual organs remain in their normal condition. There is no decubitus, and the general health is satisfactory.

The electrical phenomena in the nerves and muscles are the same as those which we have found to occur in the acute form of the disease, but are more slowly developed. The symptoms now remain stationary for a variable length of time, and then there is gradual improvement, which commences in the arms and hands, so that the patient is again able to feed himself, to write, etc. The lower extremities follow, and ultimately there may be complete recovery. Often, however, certain sets of muscles remain paralysed, so that the patient, although not quite an invalid, nevertheless remains somewhat crippled. In another set of cases the disease progresses upwards to the medulla oblongata, when articulation, mastication, deglutition, and ultimately respiration, are interfered with, and the patient sinks from exhaustion or asphyxia.

IV. *Wasting Palsy.*

The last form of disease of the anterior cornua which we have to consider, is that which is generally known as *progressive muscular atrophy*, or *wasting palsy*. This was for a long time considered to be an affection of the muscular tissue itself, unconnected with any lesions of the nervous system; and even quite recently Prof. Friedreich, of Heidelberg, has, in an able and painstaking monograph,

pronounced it to consist of a chronic multiple inflammation of the muscular fibres, which he has called *progressive chronic poly-myositis*. The microscope shows the following changes in the muscular tissue:—There is proliferation of the interstitial connective tissue of the internal perimysium, between the primitive bundles. The muscular corpuscles are swollen and increased, their nuclei proliferated, and the transverse stripes cloudy and granular. As the connective tissue continues to proliferate, the muscular fibres perish, either by simple atrophy, or after previous division, or by fatty and lardaceous degeneration. Ultimately the muscle is found to have undergone cirrhosis, and presents the appearance of a tough, thin cord, or a tendinous membrane, which only shows a few remaining insular patches of reddish muscular tissue. There is consequently considerable decrease of bulk. In a number of cases, however, myositis may become complicated, either at an early period or towards the end, with *diffuse lipomatosis*, which never commences in the muscles themselves, but always in the interstitial connective tissue, as soon as this has begun to proliferate. Fat-cells originate from the connective tissue corpuscles, which are seen to be filled with oil-globules; and these latter conglomerate so as to form regular drops of fat. When this change has set in, the bulk of the muscles appears augmented, and may increase beyond

its original size. Within this fatty mass, however, the original fibrous structure of the muscles may still be recognised by the symmetrical arrangement of the different layers of fat.

Luys was the first who discovered in this disease, in addition to the changes in the muscular tissue which I have just described, alterations of the grey matter in the centre of the cord; viz., wasting of the ganglionic cells of the anterior horns, which were replaced by granular masses containing oil-globules. These observations were confirmed by Lockhart Clarke, Jaccoud, Charcot and others. The question has therefore been much discussed whether the nature of the complaint is myotic or neurotic. Friedreich, who has with great ability contended for the myotic theory, is of opinion that progressive muscular atrophy commences as primary myositis, and may lead to secondary changes in the nervous system, which consist of neuritis, affecting first the intramuscular nerves, afterwards the nerve-trunks and the roots of the spinal nerves, and ultimately the cord itself. He would therefore consider the degenerative process in the peripheral nerves, and the ganglionic cells of the anterior horns, as simple consequences of muscular atrophy and ascending neuritis.

Although Friedreich's reasoning is ingenious, he does not explain why the nerve-roots and nerves should so often have been found healthy when the

anterior horns were diseased; but the chief objection to his theory lies in the association of this complaint with other allied diseases, which points strongly to the affection being neurotic rather than myotic. Thus it occurs together with progressive bulbar or labio-glosso-pharyngeal paralysis, which has been conclusively shown to arise from wasting of the ganglionic cells of the motor nuclei of the rhomboid fossa; and this association is easily explained by assuming the degenerative process to spread from the cord to the corresponding portion of the medulla oblongata. Again, in the later stages of locomotor ataxy muscular atrophy is noticed, showing that the disease has crept forward from the white posterior columns to the grey matter and its anterior cornua. We are therefore led to the conclusion that in all these diseases the muscular tissue is only secondarily affected, and that the symptoms depend upon those areas of the cord and medulla oblongata which become involved in the first instance, and upon those which afterwards participate in the pathological process.

Prognosis and Treatment.

Finally I must speak about the prognosis and treatment of these affections. I regret to say that both are at the present time far from satisfactory.

It is true that some few cases of infantile paralysis recover spontaneously within a month or two, and it is to these that Kennedy has given the name of "temporary paralysis;" it is also true that some few cases which are efficiently treated in the commencement recover, or at least very nearly so. Nevertheless it must be admitted that on the whole the results of treatment have, until now, been somewhat discreditable to our art. In the vast majority of cases there is ultimately permanent partial paralysis, with atrophy, deformity and contraction. As the nutritive centre of the paralysed limb is destroyed, growth is arrested; the limb becomes shorter than its fellow; the bone is thinner; the muscles waste away completely, or are replaced by fat; the arteries, nerves, and tendons become smaller and even the muscles which are not paralysed do not grow properly. All this is more marked in the upper than in the lower extremities, and more particularly in the hand and fingers. The bones are more fragile and flexible, and therefore become easily subject to deformity and fracture. Curvature of the spine is thus readily induced, as the vertebræ become too soft to be able to bear the weight of the body. Relaxation of the articular ligaments leads to complete or incomplete dislocation of the bones. Thus the knee is bent backwards and inwards, and the head of the humerus is felt to be out of its socket. Deformities of some kind are indeed rarely

absent, and various other causes besides those already mentioned contribute to their production. The degree of the paralysis is not exactly the same in the different sets of muscles in a limb, and consequently those which are only slightly paralysed may become permanently shortened by losing the influence of their antagonists. Another cause of muscular rigidity and contractions is the odd way in which paralysed children manœuvre with their limbs, more especially the legs, in moving about, when one or both legs are nearly paralysed. They walk on their hands, lean heavily on the knees and hips, and bring the body into all sorts of strange attitudes. The feet suffer more particularly, and orthopædic surgeons are aware that almost all cases of non-congenital clubfoot are owing to infantile paralysis. In the upper extremity the deformity generally spares the elbow, while the shoulder becomes raised from shortening of the deltoid, trapezius, and pectoralis muscles, and there is difficulty in moving the arm outwards; and the wrist and fingers are often permanently flexed.

What has been said about the prognosis of infantile paralysis applies, to a great extent, to the other diseases of the anterior cornua which we have been considering. The chronic form of anterior polio-myelitis in adults seems more amenable to treatment than the acute one; while for progressive muscular atrophy a really effective remedy has still to be discovered.

The treatment of infantile paralysis, which is generally recommended, consists as long as there is fever, of rest in bed, calomel, leeches, and cupping to the spine, more especially the region of the cervical and lumbar enlargement, rubbing in of mercurial ointment, and counter-irritation by tincture of iodine and blisters. With the exception of rest and a scanty diet, I believe these measures to be useless, and have therefore ceased to employ them. The only remedy in which I have any confidence, in the acute stage of this disease, is the subcutaneous injection of ergotine. Ergotine has been physiologically proved to have the power of contracting the blood-vessels of the spinal cord, more especially when it is directly introduced into the circulation, without the intervention of the stomach; and as intense hyperæmia of the arterioles and small veins, and bursting of capillary vessels of the cord from excessive distension, is one of the characteristic anatomical features of the disease; ergotine appears to be a direct antidote to that condition. I use a solution of Bonjean's ergotine in distilled water, which, if thoroughly pure, is generally not irritating; and the dose I inject is one-fourth grain for a child from one to two years of age; one-third grain for one from three to five years; half-a-grain for children from five to ten years of age; and a grain for patients upwards of ten years. These injections must be repeated according to the symptoms which may be

present, either daily or twice a day. Our guide of action in this matter should be the thermometer and the pupil. In severe cases where the temperature runs up to 103° or 104° the remedy should be used more freely than when the thermometer shows only a rise of one or two degrees. The fever being in all these cases a secondary phenomenon, consequent upon local inflammation, may be rapidly reduced by the use of the ergotine, which thus proves a truly antiphlogistic remedy; and its employment should be continued until the temperature has fallen to the normal standard. Where the pupil remains much contracted after the use of the remedy, some time may be allowed to elapse before it is again injected; but where it is large, the dose may safely be increased and repeated. The injection is not painful if well performed, and is quite as easily done, even in restless children, as it is to make them swallow a dose of medicine. The place of injection is a matter of indifference. I generally inject into the legs, as most convenient.

Such is the treatment I recommend for the attack itself. As soon, however, as the inflammation has subsided, the ergotine must be discontinued, and iodide of potassium in doses varying from two to ten grains, according to age, several times a day, be substituted for it. This serves to induce the absorption of the inflammatory effusions, to check the excessive growth of connective tissue which is

liable to follow, and thus allows those ganglionic cells, which have not been entirely destroyed by the disease, the possibility of regeneration. At the same time the constant galvanic current should now at once be used, and be made to pass through the diseased portion of the cord. If the leg only be affected, the current should be directed to the lumbar enlargement; if an arm only be paralysed, the cervical enlargement must be acted upon; and if the muscles of the body suffer likewise, the whole dorsal region of the cord should be included in the application. The electrodes should be large, the force of the current gentle, and the application be continued for from three to ten minutes, according to the extent of the lesion. It being most important that the current should pass through the anterior cornua, it is better to have one pole on the spine and the other one on the front part of the body, than to place both electrodes to the spine. Thus, if the cervical cord has to be acted upon, we place the positive pole to the nape of the neck, and the negative to the manubrium sterni; where the lumbar enlargement is diseased, the positive pole is put to the loins, and the negative a little below the umbilicus; while where the dorsal cord has to receive the voltaic influence, the positive pole should be slowly guided along the whole of the dorsal spine, the negative being left stationary at the ensiform process. Erb has recommended to send the current through the

cord, first in one direction and then in another, so as to utilise the influence of either of the poles on the diseased parts. From general considerations of the catalytic effects of the current, the action of the positive pole alone appears to me to be most called for, and I should therefore employ Erb's proceeding only if the mode of application I have just recommended should fail to effect much improvement in the patient's condition. An early resort to this mode of voltaic application is of the greatest importance, for where the ganglion cells have been either entirely destroyed by inflammation, or where those which were left uninjured or only slightly altered, have, in the course of time, become compressed and squeezed by excessive development of connective tissue, only little can be expected of any therapeutic measures. Where the case comes under treatment six months or longer after the invasion of the disease, we have to do more with the consequences of the attack than with an actively proceeding pathological process. Iodide of potassium is then useless, and small doses of phosphorus and cod liver oil are the best medicines for improving the nutrition of the nervous matter. The phosphorised cod liver oil, first prepared many years ago at my suggestion by Savory and Moore, is a very useful preparation at this stage. Subcutaneous injections of strychnia, which have been strongly recommended by Mr. Barwell, have, in my hands,

not yielded those results which would appear to have been seen by other observers; and as strychnia is a dangerous poison, and may easily do a great deal of harm, I cannot recommend its use. Mountainous or sea-air, the thermal springs of Rehme, Wildbad, Teplitz, or Gastein, and a very nutritious, even stimulating diet, are more useful. The voltaic current should likewise be applied to the seat of the disease in the cord in the way just described; but as we have at this stage to do with wasting of the paralysed muscles and with consecutive deformity, a peripheral application of the constant current and faradisation of the paralysed nerves and muscles must now be combined with it. When the nerves and muscles are found to have entirely lost their faradic excitability, faradisation is of no service; but where there is a slight faradic response, it is generally beneficial. On the whole, however, the voltaic current will be found superior to the induced, even for peripheral application, as voltaism is, in the majority of cases, the only agent which will produce any muscular response. Gymnastic exercises of the muscles, shampooing and friction of the paralysed limbs with the linimentum ammoniæ and other stimulating applications, should also not be neglected.

I cannot enter in this place into the surgical treatment of the deformities consequent upon infantile paralysis, which steps in as a last resource

when the hope of affecting a cure has vanished; but much can be done by the practitioner in preventing the development of such deformities by appropriate appliances intended to supply the loss of the muscular power.

Acute anterior myelitis of the adult, being essentially the same disease as infantile paralysis, should be treated in a similar manner. The injection of ergotine may, however, in such cases be used more freely than in children, that is to say, two or three grains may be injected at a time, more especially where there is a high degree of inflammation, as evidenced by a great rise in the temperature of the body. In chronic poliomyelitis, the internal use of the liquid extract of ergot, in doses of 20 or 30 minims several times a day, is sufficient, and should be combined with electrical applications, which in their turn may be succeeded by a course of mineral water treatment. For progressive muscular atrophy no medicine appears to be of any benefit, and the sheet anchors to which we have to trust in the treatment of that disease are galvanisation of the suffering portion of the spine, galvanisation and faradisation of the suffering muscles, and carefully-regulated gymnastic exercises of the latter.

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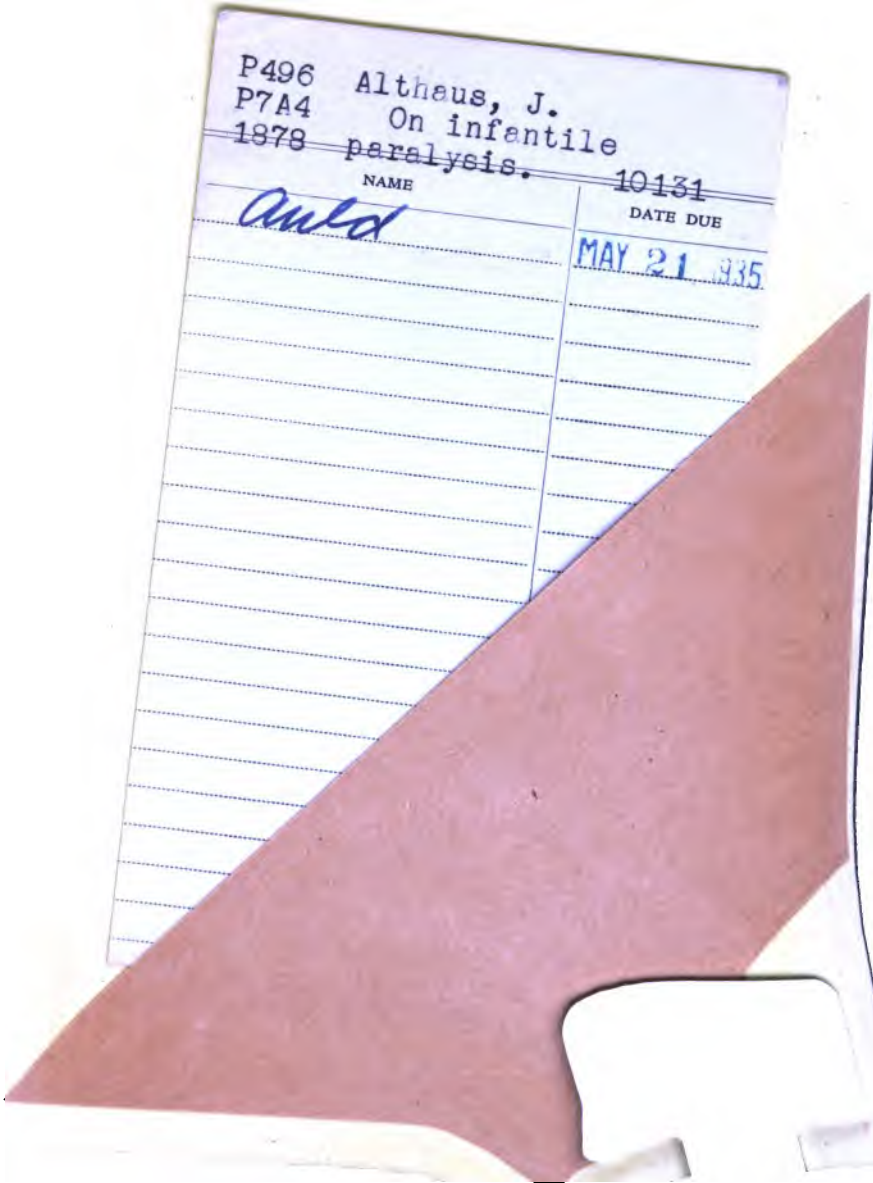
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